ORIGINAL ARTICLES

ABNORMAL BODY TEMPERATURES IN INJURIES OF THE CERVICAL SPINAL CORD.*

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The essential clinical features of the case which gave rise to this paper were a sudden, complete and unvarying paralysis and anaesthesia of all parts of the body supplied from below the level of the 4th cervical cord segment, diaphragmatic respiration, absence of the sphincter and all other body and limb reflexes except the plantar, which was at times exaggerated, transient priapism, and high temperature with slow pulse rate, all the result of a fracture dislocation in the cervical region (see paper of Dr. T. M. Williams, Journal A. M. A., July 22, 1911, p. 283, for full case report). In other words, by an unfortunate accident, there was duplicated upon a healthy young man the conditions brought about upon large dogs by Naunyn and Quincke [1] in their study of the effects of crushing injuries of the cervical cord upon variations in body temperature. The bedside record (see illustration) shows that subsequent to an initial subnormal temperature there developed a well marked hyperpyrexia which was apparently independent of external temperature, marked vasomotor disturbance, sweating, ingestion of food, and infection; was not accompanied by an increased heart rate, but was associated with rapid diaphragmatic pulmonary ventilation. Here there are presented a series of interesting physiological and clinical problems. What has suddenly destroyed that fine balance of chemical and mechanical factors concerned in the maintenance of a uniform body temperature? Is the subnormal temperature due to decreased heat production or to increased heat loss? Is the hyperpyrexia from increased heat production or diminished heat loss? If more heat is produced, can it come from the flaccid muscles or is it from the liver and alimentary tract? Is it possible that there are trophic or heat nerves associated with the muscles independent of the motor nerves? Is there an inhibitory heat center in the cervical cord or are there inhibitory fibres passing through it that are destroyed by such a lesion? Is this hyperpyrexia to be distinguished from fever due to infection? Is the whole effect produced merely that resulting from vasomotor disturbance? Clinically it is important to answer these questions. Does the occurrence of persistent subnormal or high temperature after a cord injury mean a complete transverse lesion? Can one differentiate by physical or other findings such a variation in temperature from one due to infection? Do such cases throw any light on the possibility of the occurrence of purely nervous or hysterical fevers?

Before attempting to answer some of the questions by a brief review of the great and conflicting experimental and clinical literature it might be well to call to mind that heat is produced largely by the muscles, heart, glands, intestine and liver, that according to Vierordt it is lost by warming the urine and feces (1.8%), and the expired air (3.5%), by evaporation from the lungs (7.2%) and from the skin (14.5%) and by radiation and conduction (73%) and that its regulation depends upon a careful balance of the heat produced and that lost and that the vasomotor mechanism plays an important and evident part in this control. It is also important to remember in interpreting the results of animal experimentation that the larger an animal is the greater the ratio of its volume to its surface so that a small animal has greater surface in relation to its volume, and must have proportionally more rapid metabolism and more food to keep up the same body temperature. The various types of calorimeters, the different kinds and sizes of animals experimented upon, the seasons of the year, etc., have all added factors of uncertainty to much of the work done.

Fever and heat regulation are such striking phenomena that they have attracted many scientific minds to work towards their explanation, and throughout much of the work that has been done the question of their relation to the nervous system has been ever prominent, particularly the relation of the upper cervical cord and the medulla to subnormal and elevated temperatures. As far back as 1836 Brodie [2] reported a clinical case of a crush of the cord at the level of the 5th and 6th cervical vertebrae, in which there was slow and irregular diaphragmatic breathing, weak pulse, livid countenance and prompt death with a previous temperature of 111° F. Previously in 1812 [3] he had studied the effect of urare, which paralyzes the muscle end-plates, upon temperature and the respiratory exchange, and found that less heat was generated by animals poisoned with it. In discussing his clinical case he refers to Chossat's investigations (1822) showing that division of the superior portion of the spinal cord produced remarkable elevations of animal heat. Bernard, Schiff, Bezold, Tscheschichin and others, after many experiments, largely on rabbits, obtained somewhat varying results, but in general, a fall of temperature. Naunyn and Quincke in 1869, following the idea of

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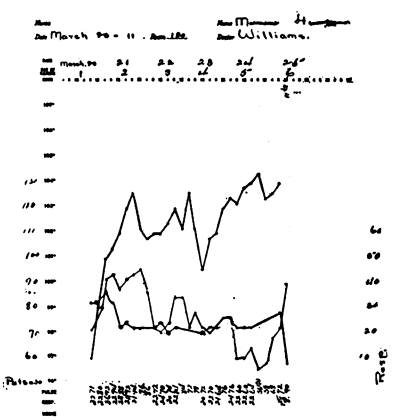
Tscheschichin who had prevented fall of temperature for a long time by keeping the animals warm after cord severance, experimented upon large dogs by crushing the cervical cord, endeavoring to produce conditions similar to clinical ones, and found that if they kept the dogs warm or in a closed chamber they usually developed high temperatures. The same result was obtained at times with simple section of the cord if made high up. These experiments were made late in the summer. Some made in winter with exposure of the animals to ordinary temperatures resulted in subnormal or normal temperatures. They considered the early fall in body temperature as due to paralysis of the vasomotors with consequent marked heat loss. This view coincides with that of Janssen.[4] The clinical cases reported by Billroth, Simon and Frerichs, in all of which high temperatures followed cervical cord injury, were thought by them to have been influenced by the fact that these accidents all occurred in the summer. In dogs a partial severance of the cord caused only slight or no elevation, and in one experiment after such a result a complete severance brought about the usual high temperature. That the lessened respiratory movements did not lead to the heat retention they were able to prove upon a dog in which they artificially interfered with respiration and yet the dog showed a lowered temperature. Their conclusion was that heat production



is increased in injuries of the 5th C. V. and they thought that instead of a hypothetical heat center in this region that fibres were severed which control oxidative processes in the body and so modify the heat production of the organs. They noted greater changes from cervical injuries than those lower down. There was an increase in heat production and in heat loss and the result obtained depended upon which factor outweighed the other. In other words after a cord lesion of the type described heat regulation ceased to exist and the body temperature became subject to various internal and external conditions.

Fischer [5] working about the same time with section of the cord at various levels concluded, after getting usually some elevation in the temperature of the dogs and rabbits with cervical section, that there was an inhibitory heat center in the cervical cord.

Binz [6] in 1870 working upon dogs with a temperature already reduced by chloroform and morphin obtained similar and even more striking results.

Rosenthal, Riegel and Parinaud obtained different results by working with rabbits and in general found that below a certain external temperature animals with severed cords did not show a rise, in fact below about 90° F., a continuous loss in body temperature. To overcome the objection to Naunyn and Quincke's work that they kept injured dogs in a closed chamber, where because of their natural inability to sweat and necessary use of the tongue and respiration to cool themselves speedy water saturation of the enclosed air took place with consequent absence of cooling evaporation, the work of Parinaud [7] was done in the open air in the summer time in Paris. He also measured the temperature of various parts of the body, an essential thing in a condition associated with a marked fall of blood pressure and probable uneven distribution of the blood. The variable results of these experiments are analogous with the variable clinical findings but nevertheless they make it perfectly clear that cervical cord severance has a marked effect on heat regulation. Wood [8] in his well known treatise on fever showed that with the fall in blood pressure associated with cord severance a marked dissipation of heat occurred if severance was above the splanchnics. Pflüger [9] showed that metabolism was reduced one-half by cutting the cord at 5 to 6 c. v. as measured by relation of O absorbed to CO_2 given off. The use of curare by Zuntz,[10] Julyet and Regnard had a similar effect and Pflüger,[11] who carefully worked out this point, found about one-third reduction in the oxygen absorbed and the CO_2 given off due to its use. Zuntz [12] also found that if curanized rabbits were kept at a constant temperature in a bath, the respiratory exchange remained constant and it was impossible to induce the prompt septic fevers that occurred in uncurarized rabbits upon injection of septic material. Various investigators, among them Gildermeister, had shown that increased CO_2 was given off by intact animals in a cold bath due to nervous reflexes stimulating metabolism. Heidenhain believed that all of these temperature effects were due to vasomotor changes and that their regulation was a function of the vasomotor system, but the experiments upon various sections of the brain, heat puncture of Aronsohn and others, etc., all show that the nervous system has, besides its vasomotor function, a heat producing one and also probably exerts, at least through inhibitory centers, a definite influence upon heat regulation. Tscheschichin, Eulenberg and Landois, and Wood and Ott all describe a rise in temperature following section of the medulla from the pons. Hitzig, Bechterew, Wood, following Eulenberg's and Landois' work, found a center in the dog's brain practically identical with that for the hind limb, the destruction of which caused variations in temperature of the contralateral side, independent of muscular action. Injuries of the corpora striatum have been found by Ott and others to result in high temperatures. The experiments of Aronsohn and Sachs [13] upon heat puncture are particularly interesting. They found (1) that destruction of the mesial side of the corpora striatum lead

to increase in temperature and that (2) electrical stimulation of this area had a similar effect. Wittkowsky [14] after heat puncture found the CO_2 content of blood unchanged, while in septic fever there was a constant decrease. Martin [15] discovered that albumen was absent in the urine after heat puncture, but usually present in fever of septic origin. Schultz [16] concluded that heat production is due to a stimulation of the muscle without visible change, at the expense of nitrogen free bodies and perhaps, is closely associated with Pflüger's "chemical tone." Rolly [17] showed that with the consumption of Nitrogen free bodies after heat puncture goes an increase in the temperature of the liver, it becoming the warmest organ of the body (Hirsch and Rolly [18]). In fact the presence of glycogen in the liver was found necessary to obtain the hyperpyrexia after puncture.

Kemp,[19] experimenting with curare, thought that he had proved that there are nerve fibres going to the muscles, distinct from the motor fibres which control the production of heat. Mosso [20] showed that after paralyzing an animal with curare the injection of strychnin led to increase temperature.

Ito [21] found that the duodenal temperature increased more after heat puncture than any other part of the body and recently (1910) Sinelnikow [22] working under the direction of Knonecker has claimed that the effect of heat puncture is upon the abdominal organs, liver, etc., rather than upon the muscles, and Streerath [23] has located the most effective site for causing elevation of temperature as a sharply bounded area at the anterior medial portion of the optic thalamus and found that strychnin produces its effects sooner, and in smaller doses on "heat puncture" animals. Pembrey [24] in 1897 reported two cases of traumatic cord section in man in the hope of clearing up the point as to which is the cause of the change in temperature; increased or diminished heat loss or increased or diminished heat production. The data furnished is insufficient, but observations were made upon deep and superficial temperatures and he concludes that the power of heat regulation is abolished by such injury and that if the patient is exposed to moderate cold the temperature falls because of increased loss of heat and diminished production of heat, in other words the normal stimuli leading to increased heat production and diminished heat loss are lacking. With heavy bed clothes increased temperature due to increased heat production, and diminished loss is obtained. He says that the paralyzed parts soon cease to sweat, the lungs ventilate less perfectly and the increase in temperature once established leads to increased metabolism.

With these experimental and clinical results in mind a brief review of the present case is of interest. In spite of the flaccid condition of all of the skeletal muscles of the body except those about the neck and head, the lessened heart action (moderate pressure with slow pulse), the absolute freedom from infection, and the small amounts of food and water taken, this patient, with a crushed cervical cord, developed a hyperpyrexia, although the weather was not unduly warm and the amount of bed clothes was very moderate. Unfortunately, because of the absence of the local weather observer we are unable to submit a temperature curve with the clinical record, but it is quite clear from the chart that the ordinary diurnal variation of the California climate played no evident part in the rise and fall of the body temperature. It is true that there was no marked vasomotor disturbance of the skin, except a persistent tasche cerebral after stroking it, only normal sweating, and that, although the diaphragmatic breathing was increased in rate, it probably was inefficient in ventilation as judged from the stasis at the pulmonary bases and the cyanosis. The mouth was usually dry and thirst was complained of. Probably some of the elevation of temperature was due to heat retention from inability of the body to cool itself by evaporation from the skin and lungs, but there was no marked interference with the loss of heat by radiation and conduction, the most important method of loss, so that we can probably look upon the hyperpyrexia as due to increased heat production. It is interesting to note that strychnin was given our patient several times and this may have played a part in increasing the temperature by some direct effect on the cord or muscles. The exact source of the heat it is impossible to determine. It certainly is most striking to get so much generated when the major portion of the body muscles are thrown out of motor function. It would seem as if chemical processes in the quiescent muscles, perhaps controlled by special nerve fibres (Kemp, Mosso), might explain it. The relation of the anterior horn cells to the muscles was not disturbed except over a limited area. There were no noticeable spasms of the neck muscles, nor evident muscular changes about the site of injury. It is true that there was a steady pull exerted upon the neck muscles by weights attached to a harness supporting the head. This may have contributed much to heat production. The liver and the intestinal tract and the various glands undoubtedly supply much of the body's heat and their part may be as great as would be indicated by the effect noted by Rolly and later Sinelnikow upon animals where no increase in temperature took place in injuries of the nervous system unless there was a preliminary store of glycogen in the liver. Unfortunately we made no glycogen determinations upon the liver in the present instance. The extent of the relation of vasomotor mechanism to the regulation of heat is difficult to determine in any given case, as well as in general. Judging from the experimental work done and our case, it would seem to play a subsidiary part in the temperature changes. It seems true, as Krehl has said, that after cord severence the body temperature becomes a "plaything of circumstances." That there are some inhibitory nerve paths that have to do with heat regulation and that pass through the cervical cord from the higher centers seems likely. It is also interesting in this connection to note that the higher the lesion the greater the effect produced upon temperature.

Clinically, the changes in temperature following cord injuries are significant in making probable the occurrence of hysterical fever as described by Dippe [25] and Levison,[26] especially where the halves

of the body differ in temperature; also of fevers in the hypnotic state and perhaps such so-called reflex fevers as catheter fever, etc. The points brought out by Wittkowsky and Martin in distinguishing the high temperatures of nervous origin from those due to sepsis are also worthy of note in differential diagnosis. Persistent high or low temperature after spinal injuries high up, where no infection is present mean, (judging from all the cases I have been able to find on record) a complete section of the cord. The higher the lesion the higher the temperature is apt to be. The damage done is usually from the preliminary crushing and haemorrhages and not from persistent bone pressure on the cord. The number of recorded cases where bone pressure persists in the cervical cord regions is very small. That the retention of the normal plantar reflex (and even its exaggeration, so that the whole leg moves in the absence of the patellar and other reflexes) may take place in complete section of the cord is a fact to be remembered, and it may indicate a state of the cord permitting of impulses reaching the muscles from the cord and having to do with heat production.

Further study of such cases as the present one may bring out important physiological facts in regard to heat regulation. It is to be regretted that conditions did not favor a more complete study of this case. Careful plethysmograph and blood pressure tracings, a study of the temperature of various parts of the body under varying conditions, an estimation of the glycogen in the liver after death would all be valuable, and not difficult to carry out. A study, by Waller's method, of the moisture given off, or the use of a calorimeter, would add much to our present knowledge. If the interest of others, who may have such cases fall into their hands, is attracted by the present report, its aim will have been fulfilled.

REFERENCES.

1. Naunyn and Quincke—Archiv f. Anat. u. Physiol., 1869, pp. 174 and 521.
2. Brodie—Med. Clin. Transactions, 1836, Vol. XX.
3. Brodie—Philosophical Transactions Royal Society, 1812.
4. Janssen—Ueber Subnormal Temperaturen, Arch. f. Klin. Med., Vol. LIII, p. 247.
5. Fischer—Centralblatt f. d. Med. Wissench, Berlin, 1869, No. 17.
6. Binz—Virchow's Archiv., Vol. LI, pp. 13-20.
7. Parinaud, Archiv. le Physiol., 2ieme serie, Vol. IV, 1877, pp. 63 and 310.
8. Wood — Fever — Smithsonian Contributions to Knowledge, No. 357.
9. Pflüger—Archiv. f. d. Gesam. Physiol., Vol. XII, 1876, p. 284.
10. Zuntz—Archiv. f. d. Gesam. Physiol., Vol. IV, p. 84; Vol. XII, p. 522.
11. Pflüger—Archiv. f. d. Gesam. Physiol., Vol. XVIII, p. 302.
12. Zuntz—Centralblatt f. d. Med., Wis., 1882, p. 562.
13. Aronsohn and Sachs—Pflüger's Archiv., Vol. XXXVII, p. 232.
14. Wittkowsky—Archiv. f. Exp. Path. u. Pharm., Bd. XXVIII, p. 283.
15. Martin—Arch. f. Exp. Path. u. Pharm., Bd. XL, 453.
16. Schultze—Arch. f. Exp. Path. u. Pharm., Vol XLIII, p. 193.
17. Rolly—Archiv. f. Klin. Med., Vol. LXXVIII, 289.
18. Hirsch and Rolly—Archiv. f., Klin. Med., Vol. LXXV. p. 307.
19. Kemp—Therapeutic Gazette, Vol. XIII, 1889, pps. 86 and 155.
20. Mosso—Virchow's Arch., Vol. CVI, pp. 80 and 126.
21. Ito—Zeitschrift für Biologie, Bd. XXXVIII, 139-1899.
22. Sinelnikow—Archiv. für Anatomie und Physiol. (Physiol. Abt.) 3 and 4, p. 279, 1910.
23. Streerath—Archiv. für Anatomie und Physiol. (Physiol. Abt.) 3 and 4, p. 295, 1910.
24. Pembrey—Journal of Physiology, 1897, Vol. XXI; British Med. Journal, 1897, Vol. II.
25. Dippe—Archiv. Klin. Med., Vol. LXIV, p. 212.
26. Levinson—Med. Record Bd., LXXVII, No. 18, 1910.

TREATMENT OF NAEVUS VASCULARIS BY THE USE OF CARBON DIOXIDE SNOW.

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Within the last ten years, or, more properly speaking, within the last five years, for it is only within that time that it has been brought into practical use, a new remedial agent has taken a definite place in dermatological therapeutics, an agent, which possesses many of the virtues of the older remedies, physical and otherwise, without many of their disadvantages; I refer to refrigeration. The principle of refrigeration, as first instituted by Detlefson [1], consisted in applying an ethyl chloride spray to the diseased areas; later liquid air was brought forward, and it was the desire for a more stable, more convenient and cheaper substitute that was responsible for the advent of solidified carbon dioxide, the credit for the utilization of which belongs to Pusey. The fact that the application of carbon dioxide or liquid air, under pressure, produces a freezing of the tissues to a considerable depth, distinguishes freezing from all other similar methods.

In order that the terms applied might be clearly understood it seems to me that it would be appropriate to classify the vascular naevi, and I would divide them, for practical purposes, as follows:

1. Flat Naevi—a. Naevus Araneus.
   b. Naevus Flammeus.
2. Hypertrophic Naevi—a. Cuticular.
   b. Subcuticular.
3. Angioma Cavernosum (Winiwater).

1a. Naevus Araneus is the ordinary spider naevus consisting of a central capillary vessel with small arborescent branches and normal skin between the branches.

1b. Naevus Flammeus consists of a superficial plexus of dilated capillary vessels, which are so closely approximated as to show no normal tissue between them, displaying a uniform discoloration, popularly known as "port-wine" mark.

2a and 2b. Hypertrophic naevi consist of a well defined, elevated, often irregular, mass of intercommunicating blood vessels of uniform color, showing a tendency to increase in size shortly after birth and then remaining stationary. The subdivisions are obvious.

3. Angioma Cavernosum is practically the same as No. 2 with the exception that it continues to increase in size at the expense of the surrounding tissues, which it destroys by pressure.

Action on the Tissues.—The initial result of the application of the snow is a temporary contraction of the blood vessels, the skin appearing white, de-